

产品名称: **Liproxstatin-1**

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生物活性:																									
<b>Description</b>	Liproxstatin-1 is a potent ferroptosis inhibitor, with IC <sub>50</sub> of approximately 38 nM.																								
<b>IC<sub>50</sub> &amp; Target</b>	EC50: 38 nM (ferroptosis)[2]																								
<b>In Vitro</b>	Liproxstatin-1 prevents BODIPY 581/591 C11 oxidation in Gpx4 <sup>-/-</sup> cells. Moreover, Liproxstatin-1 does not interfere with other classical types of cell death, such as TNF $\alpha$ -induced apoptosis and H <sub>2</sub> O <sub>2</sub> -induced necrosis, and in the bona fide L929 model of TNF $\alpha$ /zvad-induced necroptosis[1]. Liproxstatin-1 has great anti-ferroptotic activity with EC50 of appr 38 nM. Fer-1 and Liproxstatin-1 are inherently good, but not great, radical-trapping antioxidants, but they are excellent in phospholipid bilayers. Fer-1 (10 $\mu$ M) and Liproxstatin-1 (10 $\mu$ M) do not exhibit significant inhibitory activity in the 15-LOX-1 overexpressing cells, and the concentration is almost 1000-fold higher than their EC50s for subverting RSL3-induced ferroptosis in these cells (15 and 27 nM, respectively)[2].																								
<b>In Vivo</b>	Liproxstatin-1 (10 mg/kg, i.p.) suppresses ferroptosis in human cells, Gpx4 <sup>-/-</sup> kidney and in an ischaemia/reperfusion-induced tissue injury model[1].																								
<b>Solvent&amp;Solubility</b>	<p><b>In Vitro:</b></p> <p><b>DMSO : <math>\geq</math> 31 mg/mL (90.95 mM)</b></p> <p>* "<math>\geq</math>" means soluble, but saturation unknown.</p>																								
	<table border="1"> <thead> <tr> <th rowspan="2">Preparing Stock Solutions</th> <th>Solvent Mass</th> <th>1 mg</th> <th>5 mg</th> <th>10 mg</th> </tr> <tr> <th>Concentration</th> <td></td> <td></td> <td></td> </tr> </thead> <tbody> <tr> <td></td> <td>1 mM</td> <td>2.9338 mL</td> <td>14.6692 mL</td> <td>29.3384 mL</td> </tr> <tr> <td></td> <td>5 mM</td> <td>0.5868 mL</td> <td>2.9338 mL</td> <td>5.8677 mL</td> </tr> <tr> <td></td> <td>10 mM</td> <td>0.2934 mL</td> <td>1.4669 mL</td> <td>2.9338 mL</td> </tr> </tbody> </table>	Preparing Stock Solutions	Solvent Mass	1 mg	5 mg	10 mg	Concentration					1 mM	2.9338 mL	14.6692 mL	29.3384 mL		5 mM	0.5868 mL	2.9338 mL	5.8677 mL		10 mM	0.2934 mL	1.4669 mL	2.9338 mL
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<p>*请根据产品在不同溶剂中的溶解度选择合适的溶剂配制储备液: 一旦配成溶液, 请分装保存, 避免反复冻融造成的产品失效。</p> <p>储备液的保存方式和期限: -80°C, 6 months; -20°C, 1 month. -80°C 储存时, 请在 6 个月内使用, -20°C 储存时, 请在 1 个月内使用。</p> <p><b>In Vivo:</b></p> <p>请根据您的实验动物和给药方式选择适当的溶解方案。以下溶解方案都请先按照 <b>In Vitro</b> 方式配制澄清的储备液, 再依次添加助溶剂:</p> <p>——为保证实验结果的可靠性, 澄清的储备液可以根据储存条件, 适当保存; 体内实验的工作液, 建议您现用现配, 当天使用; 以下溶剂前显示的百分比是指该溶剂在您配制终溶液中的体积占比; 如在配制过程中出现沉淀、析出现象, 可以通过加热和/或超声的方式助溶</p>																									
<p>1.请依序添加每种溶剂: 10% DMSO<math>\rightarrow</math>40% PEG300 <math>\rightarrow</math>5% Tween-80 <math>\rightarrow</math> 45% saline</p> <p>Solubility: <math>\geq</math> 2.5 mg/mL (7.33 mM); Clear solution</p> <p>此方案可获得 <math>\geq</math> 2.5 mg/mL (7.33 mM, 饱和度未知) 的澄清溶液。</p> <p>以 1 mL 工作液为例, 取 100 <math>\mu</math>L 25.0 mg/mL 的澄清 DMSO 储备液加到 400 <math>\mu</math>L PEG300 中, 混合均匀, 向上述体系中加入 50 <math>\mu</math>L Tween-80, 混合均匀; 然后继续加入 450 <math>\mu</math>L 生理盐水定容至 1 mL。</p>																									

	<p>盐水水溶液中，混合均匀。</p> <p>3.请依序添加每种溶剂： 10% DMSO →90% corn oil</p> <p>Solubility: ≥ 2.5 mg/mL (7.33 mM); Clear solution</p> <p>此方案可获得 ≥ 2.5 mg/mL (7.33 mM, 饱和度未知) 的澄清溶液，此方案不适用于实验周期在半个月以上的实验。</p> <p>以 1 mL 工作液为例，取 100 μL 25.0 mg/mL 的澄清 DMSO 储备液加到 900 μL 玉米油中，混合均匀。</p>
<p><b>References</b></p>	<p>[1]. <a href="#">Friedmann Angeli JP, et al. Inactivation of the ferroptosis regulator Gpx4 triggers acute renal failure in mice. Nat Cell Biol. 2014 Dec;16(12):1180-91.</a></p> <p>[2]. <a href="#">Zilka O, et al. On the Mechanism of Cytoprotection by Ferrostatin-1 and Liproxstatin-1 and the Role of Lipid Peroxidation in Ferroptotic Cell Death. ACS Cent Sci. 2017 Mar 22;3(3):232-243</a></p>
<p><b>实验参考：</b></p>	
<p><b>Cell Assay</b></p>	<p>To induce the knockout of Gpx4, cells are seeded onto 96-well plates (1,000 cells per well) and treated with 1 μM 4-OH-tamoxifen (TAM) after plating. Cell viability is assessed at different time points after treatment (usually 72 h) using AquaBluer, unless stated otherwise, as an indicator of viable cells. Alternatively, cell death is also quantified by measuring released lactate dehydrogenase (LDH) activity using the Cytotoxicity Detection Kit. [1]</p>
<p><b>Animal Administration</b></p>	<p>Animals included in the treatment study of inducible Gpx4<sup>-/-</sup> mice are equally distributed between sex and weight, with typically 8-10 weeks of age. The average weight within the groups is between 22 and 24 g. Groups are formed to have comparable numbers of females/males of the same age. Animal weight is arranged to have a similar distribution between females and males. For the pharmacological inhibitor experiments, CreERT2;Gpx4<sup>fl/fl</sup> mice are injected on day 1 and 3 with 0.5 mg TAM dissolved in Miglyol. On day 4, compound treatment is started (Liproxstatin-1: 10 mg/kg) along with vehicle control (1% dimethylsulphoxide (DMSO) in PBS). Liproxstatin-1 and vehicle control are administered once daily by i.p. injection. Survival analysis is performed using the GraphPad Prism software and statistical analysis is done according to the log-rank test. [1]</p>
<p><b>References</b></p>	<p>[1]. <a href="#">Friedmann Angeli JP, et al. Inactivation of the ferroptosis regulator Gpx4 triggers acute renal failure in mice. Nat Cell Biol. 2014 Dec;16(12):1180-91.</a></p> <p>[2]. <a href="#">Zilka O, et al. On the Mechanism of Cytoprotection by Ferrostatin-1 and Liproxstatin-1 and the Role of Lipid Peroxidation in Ferroptotic Cell Death. ACS Cent Sci. 2017 Mar 22;3(3):232-243</a></p>